BULLETIN

OF

THE JOHNS HOPKINS HOSPITAL.

Vol. VIII.—No. 71.] BALTIMORE, FEBRUARY, 1897. [Price, 15 Cents.

CONTENTS.

	PAGE.
A Case of Dermatitis due to the x Rays. By T. C. Gilchrist, M. R. C. S., L. S. A.,	17
Lesions induced by the Action of Certain Poisons on the Nerve Cell. Study VI.—Diphtheria. By Henry J. Berkley, M. D.,	23
Puerperal Sepsis due to Infection with the Bacillus Aerogenes Capsulatus. By George W. Dobbin, M. D.,	24
Proceedings of Societies: Hospital Medical Society,	29
The Surgical Significance of Gall Stones [Dr. F. Lange].	
Correspondence: A Case of Pneumo-cardial Rupture. By Geo. S. Brown, M. D.,	33

A CASE OF DERMATITIS DUE TO THE x RAYS.

By T. C. Gilchrist, M. R. C. S., L. S. A., *Associate in Dermatology, Johns Hopkins University and Hospital.*

Since the discovery of the x rays by Röntgen, thousands of observations and experiments have been made with them both in Europe and in this country, and hundreds of investigators have exposed various portions of the body, particularly the hands, frequently, and for long periods of time, yet, after searching the literature, records of only twenty-three cases (including the present one) have been given where injurious results have followed their use, and in these, lesions of the skin only have been described. Of these twenty-three cases, twelve occurred in this country, one in Canada, four in England and six in Germany.

The first report which I have been able to find was a communication in the *Deutsche Medicinische Wochenschrift*, No. 28, 1896 (July 9th), from O. Leppin, an engineer, who had used his left hand a great deal in experimenting with the x rays. He remarked that the rays had the power of producing cutaneous lesions like sunburn. The hand presented a peculiar redness, was swollen, and a vesicular eruption appeared later on the middle and ring fingers. Where the skin was hidden by a ring it was white and quite normal. Even five weeks after discontinuing the use of the rays the altered skin gave the hand an older look than the normal one.

Professor Daniel, of Vanderbilt University, reported a case of alopecia as the result of exposure to the x rays three months previous to Leppin's communication.

After examining the other reports (the references are given at the end of the article) it was found that the eruptions of the skin presented many points of similarity, and differed chiefly in severity, according to either the length of time and frequency of exposure to the rays or to idiosyncrasy of the patient.

In Dr. Sehrwald's case, which was described fully, the lesion occurred after one exposure of forty-five minutes to the x rays, and Dr. Crocker's patient had also experienced only one exposure of one hour when an eruption appeared. In Dr. Kolle's case, the patient, a boy, was exposed once, for forty minutes, when a pronounced alopecia followed, and a similar result is recorded by Professor Daniel in a man, after one sitting; but the most interesting case of all was Professor Thomson's, of Harvard, who, being skeptical as to the deleterious results of the rays, made the only experiment which has so far been recorded, by exposing his finger to the x rays for half an hour and at one and a quarter inches from the tube, with a definite purpose of producing lesions if possible. The cutaneous trouble followed in nine days after the exposure. In two cases the patients had two sittings; in Dr. Dunn's case the first exposure was one hour, and the second, seven days later, lasted one hour and a half; and in Dr. White's case the patient had two exposures of thirty and forty-five minutes each on successive days.

In Drs. Stern's and Richardson's cases the lesions appeared after three sittings, and Dr. Skinner experienced cutaneous lesions after three or four exposures of short duration. In eleven cases the results had only followed after prolonged and constant use, the duration varying.

I append a table of the cases which I have been able to find in the literature.

Recorder.	Duration.	Distribution.	Character of the Lesions.	Subjective Symptoms.	Remarks.
O. Leppin (personal experience)..	Used very frequently for many days.	Left hand and fingers.	Peculiar redness, swollen, vesicles on middle and ring fingers.	No mention.	After five weeks hand still looks "older" than the other.
Dr. Marcuse.....	Young man, 17, exposed 5–10 minutes once or twice a day for four weeks.	Left half of face, back and chest.	Brownish redness on face, followed by desquamation; later, patch of alopecia above right ear; on back, large raw patch, exposing the corium with hemorrhagic points and exudation and a number of bullæ; on chest slighter changes.	No pain in face; on back much tenderness, but no pain was felt until just previous to the eruption.	Three months later, hair returning on the bald spot. Chest and back healed, but numerous fine cicatricial lines on the back and brown pigmentation.
Dr. Feilchenfeld	exhibited a somewhat similar case (no details given) to the Berlin Medical Society.				
Dr. Conrad.	A photographer had used the x rays very frequently and for some time.	1st and 2d fingers, left hand.	Swelling and stiffness of the joints; 1st and 2d fingers swollen and of a livid brown color on the dorsal surface from the tips to the carpo-metacarpal joints. Sense of touch greatly diminished. Mustache falling out and color was changed.	Fingers extremely sensitive and pain was of a burning and scalding character.	No mention.
Dr. Paul Fuchs (personal experience).	After prolonged use of the x rays.	Left hand and fingers.	Much swollen; skin was wrinkled, cracked and stained quite brown; condition of skin like a frozen hand; later, on further exposure, a vesicular eruption appeared.	None given.	
Dr. Sehrwald.	In a boy 13 years old. Only one exposure of 45 minutes duration. Two weeks later eruption appeared.	Abdomen.	At first hyperæmia, then a vesicular eruption. Four weeks later central portion healed, but peripheral portion covered whole abdomen. Brown color. Exfoliation soon followed.	Itching when the vesicles appeared.	Cured in 9 or 10 weeks.
S. J. R. (personal experience).	Used the rays several hours daily and eruption appeared three weeks after.	Right hand and fingers. Later left hand.	Numerous dark little blisters appeared on right fingers; then marked redness and inflammation of the skin, which later became hard, very dry and yellow like parchment. Exfoliation followed. On further exposure tips of fingers became much swollen and nails affected. A colorless, bad smelling discharge came from beneath the nails, which were gradually thrown off. Left hand was substituted and similar symptoms followed.	After inflammation hand smarted very much. Skin became insensible to the touch. After second exposure serious discomfort and pain followed.	Results similar to those of sunburn but much more acute.
Dr. H. C. Dunn.	Man aged 35 years. Exposed first for one hour and afterwards for an hour and a half, the second with interval of seven days. Two days later eruption appeared.	Abdomen.	Three hours after first exposure some nausea. After second exposure, nausea, and on second day abdomen slightly red, like sunburn; redness increased in intensity until fourth day, vesicles appeared, which developed into bullæ. Eighteen days later the patch was 7½x8¼ inches between umbilicus and chest, and was smooth, glazed and weeping for some days, notwithstanding treatment.	No subjective symptoms; neither itching nor pain; the raw surface painless and almost insensitive.	Eruption healed after 28 days, but skin was like boiled fish skin. After cauterization of surface a slough separated and a tough yellow opaque membrane formed.
Dr. G. C. Skinner (personal experience).	Exposed the wrist 10–15 minutes for 3 or 4 days in succession. Ten days later eruption followed.	Wrist.	Reddening of the skin appeared first; this increased until it was almost purple; considerable swelling followed. Desquamation followed.	Great pain and excessive tenderness.	Process of repair was very slow.
J. Macintyre (personal experience).	Worked for months with the x rays before cutaneous lesions were produced.	Hand.	Appearance of a sunburn. Skin red and swollen, followed by exfoliation of the "epidermis" and loss of hair.		Author thinks condition due to heat and electricity; the latter the chief cause of injury to the tissues.
Dr. Freund.	10 days, child who had hypertrichosis.	Neck, back, upper arm and scalp.	x rays used as a depilatory for 10 days, when the hair began to fall out.	None given.	

Recorder.	Duration.	Distribution.	Character of Lesions.	Subjective Symptoms.	Remarks.
Dr. E. E. King.	Patient used the *x* rays for two and a half months from two to six hours daily before cutaneous lesion appeared.	Hand, left.	Hand felt stiff, then became swollen and large "blisters" formed; after five weeks rest he exposed the hand again 7–8 hours daily. In two weeks left hand became swollen, tender, discolored; vesicles followed, and the finger-nails began to fall off. The hairs disappeared. Left side of the face was also affected. The skin was swollen and erythematous, and the hair over the temple disappeared. Eyebrows were almost gone and left side of the mustache had almost disappeared.	Great pain followed the appearance of the blisters. Second eruption followed by aching. Face became tender and painful.	The case has been carefully and continuously watched.
Dr. M. J. Stern.	Patient was exposed at three sittings of 40–50 minutes duration. Eruption appeared two days afterward.	Extended from the side of the face down to the umbilicus.	Erythematous blush first appeared, which a few days later appeared like a severe burn, followed by a large slough.		Examination of the chest after a gun-shot wound.
Dr. F. Kolle.	A boy 12 years old. Whole body exposed 40 minutes. Sixteen days later alopecia appeared.	Right side of head.	Large area of alopecia over the region exposed to the rays. Hair fell out all at once the night previous. Skin slightly œdematous.	No symptoms.	Later, lanugo hairs began to appear.
Dr. J. C. White.	Young lady exposed to the rays 30 minutes on one day and 45 minutes on the next day. Eruption appeared the following day.	Sternal region.	Skin was first reddened, then blistered. Three months later the patch showed angry-looking granulations which had refused to heal.	Granulation surface very sensitive and the seat of severe neuralgic pain.	At the time of writing there were still two open spots.
Prof. E. Thomson.	Exposed half an hour at a distance of one and one-half inches from the tube. Eruption appeared nine days after.	Little finger of left hand.	Finger became hypersensitive, then dark red, swollen, stiff, and soon after vesiculation occurred. Pus formed and the epidermis became detached, but pustules remained over 3 weeks.	Pain.	
Dr. H. R. Crocker.	Boy aged 16. One exposure of one hour. Eruption appeared next day.	Epigastrium.	A Crookes tube placed five inches distant—flanelette shirt intervened. Next day, skin deep red in color. Nine days later vesicles appeared. Palm-sized patch, well-defined, purplish red. Vesicles increased in number; 9 days later the epidermis had separated. Healing very slow. Alopecia very slight.	Stiffness felt at first and soreness when the vesicles appeared.	
Prof. John Daniel.	Man; exposure 1 hour. Alopecia 21 days later.	One side of scalp.	Tube was 1½ inches distant from head. Bald spot was 2 inches in diameter; skin healthy.	None.	
Dr. M. H. Richardson.	Woman. Exposed three times to the rays, 20, 30 and 35 minutes. Eruption appeared two days later.	Abdominal region, particularly over liver area.	Bulb was 18 inches distant. First appearance was that of sunburn, gradually became brown, and ulcer, 8 inches in diameter, formed; slough thrown off; very slow in healing.	Pain accompanied the first appearance of the lesion.	Four months later, even after curetting, granulating surface 4 inches in diameter still remained.
Dr. W. E. Parker.	Man. Exposed five times, from 20 minutes to 80 minutes.	Jaw and neck.	After two exposures face and neck became swollen and red. The later sittings were also followed by eruption.		16 days afterwards skin discolored and exfoliated.
University of Minnesota.	Man whose ear was examined for several hours.	Ear and surrounding scalp.	The parts presented later a frozen appearance. All the hair from this side of scalp was lost.	No pain or sensation at the time.	
Dr. Banister, U. S. Army.	Man. Numerous exposures.	Abdomen, chest and beard.	At first a patch, red, inflamed, hyperæsthetic, about half the size of a man's hand. The lesion increased in size until a denuded surface 8x15 inches was formed. Exudation profuse and uncontrollable. Beard turned white in places and growth stopped.	Raw surface, very painful and hyperæsthetic. Pain agonizing even six weeks after.	Patient confined to bed for one month, and will probably be in bed another month.
Dr. T. C. Gilchrist.	*x* ray operator. Eruption began after frequent and continued exposures.	Right hand, wrist, and lower portion of forearm dorsal surface.	Hyperæmia and swelling of back of hand; afterwards inflammation of fingers and hand. Skin became dark brown in color and gradually exfoliated.	No pain at first, but afterwards severe aching and shooting pains, which lasted about 2 weeks.*	

* February 5, 1897, patient writes, "My hand is much about the same. I cannot use those (first) three fingers much better; and my nails are coming off, that is, the old nail is like a shingle on top of the new one."

As far as the cutaneous lesions were concerned, the skin of the trunk appeared to suffer the most severely, although when the hand was attacked the pain was the most prominent feature.

As to the character of the lesions produced, the majority of observers describe a peculiar redness or erythema at first, then swelling of the derma, followed by a deep discoloration of the skin until it became quite a dark brown color. Exfoliation of the pigmented skin followed later. In a number of cases vesiculation occurred after the hyperæmia, and quite a serious eruption resulted in two cases after only two exposures. The most severe effects followed after exposing the same region again to the rays when the lesion had only partially or wholly healed.

The cases of Dr. Dunn, Dr. Banister and S. J. R. showed perhaps the most severe results. Other lesions produced were alopecia, loss of the finger-nails, and obstinate ulceration. In connection with the subjective symptoms, severe pain and aching were present in the majority of the cases, especially with the eruptions on the hands. The lesions on the abdomen and back were, rather curiously enough, unaccompanied by pain.

Many theories have been advanced to explain the injurious lesions produced by the *x* rays. Some have supposed them to be entirely due to frequent and long exposures; but that is not tenable, because in five cases (Dr. Sehrwald's, Dr. Kolle's, Dr. Crocker's, Professor Daniel's and Professor Thomson's) marked results followed a single exposure, and in two cases two sittings only preceded the eruptive phenomena, and finally three other cases experienced deleterious results after only three exposures. Others again have compared all the phenomena to severe sunburn, but it can be demonstrated that more severe results have followed the use of the rays than have ever been known from exposure to the sun. Tesla believes that the hurtful action of the *x* rays is not due to the rays but to the ozone generated in contact with the skin. Ozone, he says, when abundantly produced, attacks the cutaneous surface, its action being no doubt heightened by the heat and moisture of the skin. In his discussion of the rays themselves he speaks of the disintegration of the electrodes, especially if they are of aluminum, but this is so slow that no appreciable diminution of the weight results, even after long use; it follows, he says, that the matter conveyed by the Röntgen stream is so minute as to escape detection. Some bulbs which he has used for a number of months showed that the bombarded spot of the glass was entirely permeated with particles of the aluminum electrode, but it would probably require years of constant use to accumulate any appreciable amount of matter outside. He further remarks "that no experimenter need be deterred from carrying on an investigation of the rays for fear of poisonous or generally deleterious action, for it seems reasonable to conclude that it would take centuries to accumulate enough of such matter to interfere seriously with the process of life of a person." His last remark is certainly incorrect. His ozone theory is also incorrect, as I shall show later.

Professor Thomson, with the definite idea of producing cutaneous lesions, exposed his finger to a bulb of low potential and used a small 24-plate static machine. "The rays came from a bombarded spot, and were limited to the area which the Röntgen rays could reach. The tube was of blue glass, with a clear German glass window from 1½ to 1¾ inches in diameter opposite the bombarded spot." One striking feature was the fact that the fingers opposite the blue glass were not affected, as this was so dense as to absorb the rays. Only where the little finger was opposite the clear glass was it affected with a sharp line of demarcation. He says the blue glass would have been transparent to the ultra-violet rays. He also thinks that brush discharges have nothing to do with the cutaneous phenomena. His theory is that the deleterious effects are due to the *x* rays themselves or something that goes with them.

The case which I will now report presents a sufficient number of interesting points to make it worthy of record.

The patient is a healthy-looking man, 32 years of age, who came to me November 18, 1896, with the following history: During the first week in September he exposed his right hand, wrist and lower portion of the forearm to the *x* rays while exhibiting an apparatus, each exposure lasting two or three minutes. No bad results followed, but on October 1st he came to Baltimore and resumed the use of the *x* rays for the same purposes. After he had exposed his hand for three weeks for four hours daily he noticed that the skin of the back of the hand, wrist and forearm began to turn very red and became "puffed up," although he suffered no pain. The swelling first occurred on the back of the hand from the knuckles to the wrist, then "inflammation set in" and he was compelled to stop his demonstrations. From about October 21st the hand, wrist and lower fourth of the forearm gradually became more inflamed and swollen, and the lesions spread to the fingers. The affected area ached and throbbed so much that he frequently could not sleep at night; there were also shooting pains which gradually increased in intensity and extended up the forearm along the ulnar side. These symptoms continued for a week when the patient consulted Professor Chambers, of Baltimore, who advised him to bathe the hand frequently in hot water and ordered bromides internally with benefit. The symptoms were much relieved and the swelling had gone down considerably in two weeks time. Three weeks after he had first sought medical advice he consulted me about the diseased condition of the skin.

Condition at the time of the first examination. The right hand on its dorsal surface presented a deeply pigmented condition. The skin over the dorsal surface of the fingers, hand and wrist was of a very dark brown color, and part of it was already exfoliating. The skin was dry, infiltrated and wrinkled. The patient says the skin was more of a greenish hue at the end of the first week after it became affected. In places near the lateral margins of the hand a slightly vesicular appearance was presented. They were not true vesicles, but were due to the loosening of the surface of the epidermis, and contained only air. The pigmented skin could very easily be peeled off from the hand without any pain, leaving a dull, dry, reddish surface beneath.

On comparing the two hands they were found to be practically of the same temperature. The fingers of both were of about the same calibre. A photograph, which is represented here (Fig. 1), was taken of the hand the first time I saw him,

and it shows very well the exfoliation of the epidermis. It also represents the position of the hand which was most restful and least painful to the patient. The palmar surface was dry and paler than that of the normal hand, but the palmar surface of the fingers appeared swollen.

I saw the patient ten days later, and the exfoliating epidermis had all been removed; the skin now presented a glossy appearance, and over the fingers it appeared to be tighter than on the corresponding fingers of the healthy hand. The palmar surface was still a little drier.

On careful examination the patient complained of rather severe pain when the first phalanx of his right index finger was grasped, and it was then noticed that this bone was distinctly thickened, especially as compared with the corresponding phalanx of the other hand. The first and second phalanges of all the fingers were found to be thickened, but the increase in size was most marked in the first phalanges of the index and second fingers. Further careful examination of the other bones of the hand revealed a very painful spot over the wrist joint. The metacarpals were also tender on pressure, and the head of the second metacarpal was enlarged. The hairs were found to be less numerous on the affected hand, but they may have been removed in tearing off the exfoliating epidermis.

Movements of the hands. All movements were found to be quite difficult of accomplishment and very painful. When the patient first consulted me, voluntary movements of both fingers and hand were abolished, except of the little finger, which could be slightly flexed. Wrist movements were also very limited in extent and caused much pain. The thumb and fingers could scarcely be apposed. The patient could not pick up a lead pencil or penholder, and could grasp with but slight force; if he could get his finger or thumb beneath an object he could pick it up. He complained of the joints being very stiff.

Sensation was very much impaired before exfoliation occurred, and even after that it was much diminished as compared with the normal hand. The sense of touch on the palmar surface was also markedly decreased.

When the distinct thickening of the phalanges was detected I requested him to obtain careful *x* ray photographs of both hands under similar conditions at the same distance from the bulb. The accompanying photographs show the bones of the two hands clearly, and I am particularly indebted to Professor W. C. A. Hammel, of Baltimore, for such excellent pictures.

The hands were placed exactly twelve and a half inches from the *x* ray tube, the distance being carefully measured from a plane passing through the long diameter of the tube to the fixed support on which the hand rested. The time of exposure was exactly three minutes for each hand.

It will at once be noticed from the photographs how much thicker all the first phalanges of the right hand (Fig. 3) are than those of the left normal hand (Fig. 2). The increase in size is particularly marked in the first phalanges of the index and second finger; that of the little finger is less marked. The second row of phalanges is also thicker than normal, but less thick than those of the first row. The heads of the second and third metacarpal bones are enlarged, and their shafts are also slightly thickened. It cannot be said, from the photographs alone, that any of the carpal bones of the affected hand were at all increased in size.

A number of other points were discovered from these photographs. The spaces between the bones at the joints, particularly at the metacarpo-phalangeal and the interphalangeal joints, were much less marked and narrower than in those of the normal hand. The outline of the affected bones was also more irregular and rougher than that of the left hand. On looking at the hand as a whole, it is also, I think, noticed that all the bones are denser in appearance in the affected hand.*

Two portions of skin were excised for histological purposes on the first day. One portion was taken from dorsal surface of the phalangeal region of the third finger, and the other from the lateral margin of the hand over the base of the metacarpal of the little finger. Neither stained nor unstained sections demonstrated the presence of any foreign particles, and only showed chronic inflammatory changes. The horny layer was thickened and half of it was partially detached. A large number of brown pigment granules were found in the exfoliating portion. The mucous layer was not thickened, but it was more pigmented than normal. In the corium the vessels were dilated, and the pigment cells of the papillæ were almost as numerous as are usually found in a section of negro skin. It was suggested that particles of platinum might have passed from the tube through the glass bulb deep into the tissues.

Portions of the exfoliating skin were accordingly submitted to Professor Abel for chemical analysis, and he has very kindly furnished me with the following brief report: "I could find no platinum in the pieces of epidermis that you left with me for analysis. These pieces weighed, just as you sent them, 640 milligrams, or about ten grains."

Summary. In reviewing this case, there is here an example of a disease of the skin which was brought about by frequent and prolonged exposure to the *x* rays. The lesions, as I have already described, were first those of hyperæmia, then œdema, without the accompaniment of pain. Subjective symptoms were felt later, and continued to increase in severity (he was still using his hand for exhibition purposes) until, on account of the severe pain, he was compelled to stop his work. These subjective symptoms consisted of aching, throbbing and shooting pains. The color of the skin gradually changed to a deep brown, and the epidermis began to exfoliate. The bones of the hand were very tender on pressure, particularly the first phalanges of the index and second finger and the carpal bones. The movements of the hand became so limited that it was practically useless for some weeks. Sensation was also much impaired, but after exfoliation occurred it recovered again, but only gradually. Recovery of all the lesions has been very gradual indeed.

The photographs have revealed what has never been observed before, viz. a distinct osteoplastic periostitis, and probably an osteitis, particularly of the first and second rows of phalanges of the index and second fingers, also of the heads of meta-

*As an interesting point the photographs show the outlines even of the finger-nails and the interossei muscles.

carpal bones of the same fingers, and judging from the symptoms, even of some of the carpal bones.

This then accounts for the severe symptoms, the aching, throbbing and shooting pain which prevented sleep. The density of these bones has also been increased, showing that even bone tissue has been affected.

A complete demonstration is thus afforded of the powerful, piercing character of the *x* rays, and the severe, painful symptoms which have been described by other observers are probably due to the inflammation of the periosteum, and possibly the bone, besides the softer tissues.

This inflammation has also extended into the joints, which would explain the loss of movements, and pain, when they could be used later.

As the result of these observations, it proves that the *x* rays are even more powerful than have been generally thought, that the deleterious effects can in some cases be quite serious, and that the cutaneous manifestations are not the most severe of the lesions, but those of the deeper tissues, and particularly of periosteum and bones, being more severe.

The discovery of this deeper and more profound effect at once overthrows many of the explanations which have been advanced to account for the cutaneous lesions. Tesla's theory of the ozone generated in contact with the skin will not explain these deeper effects. Prof. Thomson has demonstrated that they are not due to the ultra-violet rays. They cannot be compared to sunburn, since no case, as far as is at present known, has been published where sunburn has produced periostitis and even osteitis. Nor can such serious results be produced by brush discharges, and Thomson has also proved that these cannot be the causes. His further explanation that the effects (he was not aware of the deeper injuries to the soft tissues and bone) are due to the *x* rays themselves or something which accompanies them, is rather indefinite. It was suggested to me, as I have already mentioned, that it might be due to the platinum particles piercing the bulb and then attacking the tissues, but this almost appears impossible, since serious cutaneous effects have even followed only one exposure of half an hour (*vide* Thomson's case).

I consulted with Professor Ames, Associate Professor of Physics in the Johns Hopkins University, who, after reviewing all the facts of the case, kindly wrote as follows concerning the present theory of the *x* rays:

"The radiation in an '*x* ray tube' may be divided provisionally into three classes: ether-waves, which may have wave-lengths from 150 to 800 $\mu\mu$, approximately; kathode rays, which undoubtedly are streams of matter, electrically charged; *x* rays, about whose nature there is no conclusive evidence at the present time. If the walls of the tube are thin enough and of suitable material, all these radiations will emerge and pass into the surrounding air. It is a matter of doubt if the kathode rays observed outside the vacuum-tube are the same as those inside; but the inner ones undoubtedly cause the outer ones. There is no evidence that *x* rays carry with them particles of matter, or that they directly cause a stream of particles; in fact, all known facts seem to point to the belief that they are ether-waves of extreme shortness."

It will thus be seen that the opinion expressed here does not make it possible for the *x* rays themselves to produce such deleterious effects as have already been described, but Dr. Ames mentions the fact that the kathode rays are undoubtedly streams of matter electrically charged. Here then we have some possible grounds for the theory that the lesions may be due to the entrance of particles (platinum in our case) into the injured tissues, and that the kathode rays which accompany the *x* rays may be the cause of the trouble, and not the *x* rays themselves.

On clinical grounds there is considerable support for this, at first sight, improbable theory. If the lesion extends at all deeply, it leads to the formation of ulcers, which are extremely intractable, and they may be due to irritating particles still present in the tissues.

I do not think that the possibility of injury ought to deter one from using these wonderful rays in surgical work, because only a few have been affected out of thousands who have been exposed to them. By keeping, as Thomson says, some distance away from the rays, injurious effects will hardly follow their use, and when the exposure is for a short time, unless, as may happen in all other diseases, idiosyncrasy plays a prominent part.

When I viewed my own hand two or three times near a new Edison bulb, through a tungstate of calcium screen, after four or five minutes I distinctly felt a tingling sensation throughout the dorsum of the hand; this symptom lasted for ten or fifteen minutes and passed away without any further results. It occurred to me that *x* ray operators and experimenters should expose to the rays the palmar surface of the hand, which is protected by a much thicker horny layer, rather than the back of the hand, which is much less protected.

In conclusion I would strongly advise all *x* ray operators and experimenters who develop any special idiosyncrasy, to abstain from their use if they find that the slightest deleterious results follow an exposure to them.

Bibliography.

John Daniel: Medical Record, April 25, 1896, vol. 49, No. 17.

O. Leppin: Deutsche medicin. Wochensch., No. 28 (July 9), 1896, p. 454.

Marcuse: Deutsche medicin. Wochensch., No. 30 (July 23), 1896, and No. 42 (October 15), 1896; also abstract in Brit. Med. Journal, Aug. 15, 1896.

Feilchenfeld: Deutsche medicin. Wochensch., No. 30 (July 23), 1896.

Conrad: Codex Medicus, August, 1896.

Paul Fuchs: Deutsche medicin. Wochensch., No. 35 (Aug. 27), 1896, p. 569.

Sehrwald: Deutsche medicin. Wochensch., No. 41 (Oct. 8), 1896; abstract in Brit. Med. Journal, Nov. 7, 1896.

S. J. R.: Nature, No. 1409, vol. 54, Oct. 29, 1896.

H. C. Dunn: Brit. Med. Journal, Nov. 7, 1896.

G. C. Skinner: The Journal of the American Medical Association, No. 20, vol. XXVII, Nov. 14, 1896.

Macintyre: Nature, No. 1412, vol. 55, Nov. 19, 1896.

E. E. King: Canadian Practitioner, Nov. 1896.

FIG. 1.

Case of *x* ray dermatitis. Photograph showing exfoliation of the epidermis.

FIG. 2.

x ray photograph of left hand (normal).

FIG. 3.

x ray photograph of the right hand showing thickening of some of the bones.

The Friedenwald Co., Engravers and Printers.

M. J. Stern: The American Medico-Surgical Bulletin, Nov. 21, 1896.

F. Kolle: Brooklyn Medical Journal, December, 1896, vol. X, No. 12.

J. C. White: Boston Medical and Surgical Journal, Dec. 3, 1896, vol. CXXXV, No. 23.

E. Thomson: Boston Medical and Surgical Journal, Dec. 10, 1896, vol. CXXXV, No. 24.

Nikola Tesla: Abstract in Public Opinion, vol. XXI, No. 24.

M. H. Richardson: The Medical News, Dec. 26, 1896, vol. LXIX, No. 1250.

The Medical Record: References to case at University of Minnesota, vol. 50, No. 25, Dec. 19, 1896.

W. E. Parker: New Orleans Medical Journal, Sept. 1896, vol. XLIX, No. 3.

H. R. Crocker: Brit. Med. Journal, Jan. 2, 1897, No. 1879.

W. B. Banister: Medical Record, vol. LII, No. 4, Jan. 23, 1897.

Freund: Münch. Med. Woch., No. 3, Jahrg. 44, Jan. 19, 1897.

LESIONS INDUCED BY THE ACTION OF CERTAIN POISONS ON THE NERVE CELL.

STUDY VI.—DIPHTHERIA.

BY HENRY J. BERKLEY, M. D.

During the course of this investigation, an article by Ceni* on the same subject came into my hands, and the results determined, by both investigations are so entirely similar that it seems hardly necessary to repeat in full the details of the study.

Ceni's work was done on rabbits and horses, with an acute and a chronic series. The first, after the inoculations with diphtheria cultures, lived from three to four days, and showed, in exquisite form, the beginning of the moniliform swellings of the dendrites of the cortical and hippocampal cells common to the action of all the toxins of the infectious series. The chronic series lived from 36 to 41 days after the inoculation with the toxin, during which period they lost full half their weight. Ceni also utilized the cerebra of horses which had been used in the Milan Bacteriological Laboratory to produce diphtheria serum. In these, after three or four months of the treatment, he found the cortical cells far degenerated, and to a degree not seen with the rabbits, the stouter dendrites and corpora of the cells being extensively involved in the destructive process.

Interesting to note, he also finds distinct lesions of the vascular neuroglia, principally tumefaction of the bodies and branches, the one attached to the blood-vessels' walls showing the alterations most distinctly, all being identical with those described and recorded in the III. study of this series (Art. Ricin Poisoning).

Our present series consisted of five diphtheria guinea-pigs, whose brains had been sent me by Dr. Flexner for the purposes of the study. The first two brains, after hardening and examination of the sections, were rejected on account of the possible presence of post-mortem changes. The other three cerebra were carefully hardened in Müller's fluid and treated afterwards by the silver phospho-molybdate formula.

Guinea-pig No. 3 received an injection of the toxin at 2.30 P. M. and died about 3 A. M. the following morning. No. 4 was inoculated at 2 P. M., was re-inoculated two days later at 10.30 A. M., and died two days afterwards at noon. No. 5 went through the same course of treatment as No. 4 and died approximately at the same hour.

The local manifestations at the site of the inoculation were experimental membranes, œdema, and slight central necrosis. The lymph glands throughout the body were enlarged, there were small hemorrhages in the mucous membranes, and ecchymoses and necroses of the abdominal organs, with considerable fluid in both the thoracic and abdominal cavities.

With Case 3 the dendrites only show beginning changes. There are numerous small moniliform swellings scattered over their branches, not focally but uniformly throughout the cortex, and over the sites of these tumefactions the gemmules are either lost or are very sparingly present, while on those that have normal dendrites they are undiminished in numbers. On the stretch of dendrite between the swellings, which either appears normal or slightly below the natural calibre, the lateral buds are beginning to disappear, as shown by the paucity of their numbers and lessened tendency to assume a deep black tint. The bodies of the cells do not appear to be in any way changed in staining properties, or to have lost in any degree the angularity of their contours.

The vascular neuroglia cells are uniformly stained throughout the outer layers of the cortex, and have their tentacles, particularly the vascular foot, somewhat swollen, the degree being considerably less than in some specimens we have noted after the injection of other toxins. The axis-cylinders are distinct, and show no alterations. The terminal apparatus of the cortical fibres were so infrequently stained that it is hardly possible to determine with certainty whether they were pathologically altered or no, but those found appeared to be normal.

The larger vessels showed neither thickenings nor ruptures of their coats.

With cases Nos. 4 and 5 the pathological alteration is similar to that in No. 3, only increased in intensity, from the longer duration of the poisoning, and, perhaps, also from the repetition of the inoculations. The moniliform nodules are well marked over long stretches of cells in the pyramidal region of the sections, and are uniformly larger than in the more acute case. Also the denudation of the gemmules is

* Carlo Ceni: Riforma Medica, vol. I, 1896.

marked by the greater absence of these pin-like projections from the dendrites.

Some of the psychical cells are beginning to show alterations of their outlines, appearing now rounded in contrast to the third case.

The vascular neuroglia also shows signs of more advanced swelling, the tentacles being thicker and the pseudopodia more prominent.

The axôns and collaterals are well stained, but show no unusual varicosity or other departure from the normal. A few of the cells having upturned axôns are well stained, but the morbid changes are of less intensity than with the pyramidal variety.

The cerebellar cells, as in the previous studies, showed less damage than those of the cerebrum. The Purkinje variety was the only one recognisably involved. The peculiar axôns of the basket cells were natural, and the various nerve fibres throughout the outer lamina of the cortex retained their histological appearance.

From the studies conducted on the cerebra of hydrophobic and diphtheritic guinea-pigs it would appear that all severe infections were followed by brain-cell degeneration of an order not dissimilar to that found in the cells of the abdominal viscera under similar conditions, a degenerative process unaccompanied by inflammatory reaction and a tendency to atrophy and necrosis.

PUERPERAL SEPSIS DUE TO INFECTION WITH THE BACILLUS AEROGENES CAPSULATUS.

By George W. Dobbin, M. D., *Resident Obstetrician, The Johns Hopkins Hospital.*

The following case, which occurred in the Out-patient Obstetrical Department of the Johns Hopkins Hospital, is interesting from the fact that we have been able to find reports of only a few similar cases in the literature.

We were called to see the patient, Paulina A., about eleven o'clock on the evening of March second, the messenger saying that she had been in labor about two days and the midwife was unable to deliver the child.

As the patient and midwife were Polish it was exceedingly difficult to obtain an accurate subjective history of the case, but after considerable difficulty it was found that she had had an abdominal operation done a year ago in Germany, the nature of which we were unable to ascertain. She was at the end of her third pregnancy, and had been in labor two days.

Examination showed her to be a person of medium size, of an exceedingly pinched and pale appearance about the face, with weak and rapid pulse, running from 130 to 140 beats per minute.

The abdomen was enlarged to about the size of a full-term pregnancy. Just above the symphysis was the pigmented cicatrix of the abdominal operation referred to. The fundus of the uterus presents the bullet-shaped appearance suggestive of pelvic contraction; it reaches to a point midway between the umbilicus and xyphoid cartilage, and was in a condition of tetanic contraction. Palpation showed a large child in the left-occipito-iliac-anterior position, with the head impacted in the pelvis. The fœtal heart could not be heard, and the patient was not having regular labor pains.

Measurements of the pelvis showed that she had a generally contracted pelvis, with a *conjugata vera* of approximately 8 cm.; the other measurements were as follows: distance between iliac spines 22.5 cm., between iliac crests 25 cm., between trochanters 28 cm., Baudeloque diameter 17 cm., and a diagonal conjugate of 10.5 cm.

By vaginal examination we found that the vaginal outlet was much relaxed. There was a very large caput succedaneum presenting in the vagina. The head was very firmly impacted in the pelvis and could not be pushed up. The sagittal suture was in the transverse diameter of the pelvis, with the small fontanelle to the left. The impaction of the head was so great that the cranial bones overlapped one another to the extent of one centimeter.

On approaching the bed a very sweetish, offensive odor became noticeable, and a distinct bubbling, crackling sound could be heard. This was thought at first to be due to the escape of gas from the rectum, but on turning down the bedclothes it was seen to come from the vulva, which was bathed in a dirty reddish-brown fluid containing many gas-bubbles. The bubbling, crackling sound was continuous, and was much more apparent when the patient's genitals were exposed.

Diagnosis. Generally contracted pelvis, with *conjugata vera* of about 8 cm.; large child in left occipito-iliac anterior position, with head impacted in the pelvis; arrest of labor.

Owing to the mother's condition, and as the child was already dead, immediate delivery was indicated. She was given chloroform, placed across the bed in the lithotomy position, and a Tarnier axis-traction forceps was applied by Dr. Williams. The head was brought down to the outlet with great difficulty, and as it became more and more crushed out of shape with each traction it allowed the forceps blades to slip several times. The lateral blades of the Tarnier basiotribe were then applied, screwed tightly together, and the crushed head delivered.

Tractions now made on the delivered head showed that the shoulders were so firmly impacted in the pelvis that they could not be delivered without tearing the head from the body. The blunt hook was then put into the posterior axilla and that shoulder brought down; this manipulation caused the separation of the epiphysis from the shaft of the humerus, and in order to effect delivery of the trunk it was necessary to put the blunt hook between the ribs beneath the substernal notch and literally drag the child from the mother.

Just as the child was delivered there was an escape of gas from the uterine cavity accompanied by an explosive sound. The gas possessed a most sickening fœtid odor, which was so intense that Dr. Williams was obliged to leave the patient and seek the outside air.

The third stage of labor also gave considerable difficulty, for

the uterus having been for so long in a tetanic condition, refused to retract, and it was finally necessary to deliver the placenta manually.

Retraction still being very slow, a hot intra-uterine douche of boiled water was given, which had, however, little effect; but there being no excessive hemorrhage, the patient was put back to bed. Her condition, while very bad, was not much worse than at the beginning of the operation. She was given strychnia, brandy, etc., and left for the night.

March 3rd, 9 A. M. The patient was seen by the nurse, who reported her condition in no way improved. She had repeated attacks of vomiting during the night., Temperature 98.4°, pulse 144, irregular, weak and intermittent.

5 P. M. The patient was decidedly worse than in the morning. Temperature 101.2°, pulse 144, weak, irregular and intermittent, her face presented an anxious expression, and the respirations were rapid and irregular.

During the morning "gas bacilli" had been found in the tissues of the fœtus and placenta, so a fatal prognosis was given to her husband. At this time in no part of the body could we detect the presence of emphysema. Slips and cultures were made from the lochia, which showed staphylococci, streptococci and large numbers of gas bacilli.

As well as we have been able to find this is the first instance in which a diagnosis of this form of puerperal infection has been made before death.

10 P. M. The patient is decidedly worse, no gas detected in the tissues. The patient died three hours later, and soon afterwards the body began to swell.

March 4th, 10 A. M. Seven hours after death. The body was enormously swollen, being almost twice its original size, the face was swollen entirely beyond recognition, from the mouth and nose there exuded a frothy, bloody serum, and from time to time large bubbles of gas could be seen to escape. The abdomen was enormously distended and gave a deep drum-like note on percussion. Over the entire body we could obtain the characteristic crackling of gas in the subcutaneous tissues.

The husband unfortunately allowed no autopsy.

Pathological report. The fœtus and placenta were brought back to the laboratory and the following is a copy of the pathological report:

The child weighs 2900 grammes, without the brain; its length is 55 cm. The head is entirely crushed, the right arm almost completely torn from the body, and there is a large jagged wound in the chest, all of which were caused by the craniotomy instruments and blunt hook.

The fœtus is very offensive, being much decomposed and macerated. The entire body is emphysematous, and everywhere, on making pressure, we obtain the characteristic "feel" of gas in the subcutaneous tissue. By making pressure on the chest, large bubbles of gas are seen to escape from the wound in the neck. The body was then put under water, and on puncturing the abdomen there was marked escape of gas, which when ignited shot up in a flame six inches high.

Cover-slips made from the blood and subcutaneous tissues show large numbers of bacilli morphologically similar to the "gas bacillus" of Welch and Nuttall. Cultures on sugar media gave the characteristic growth.

The placenta is of a distinctly bilobed shape, dimensions being 18x14x1.5 cm. and 10x9x1.5 cm. for the greater and lesser lobes respectively. Its weight is 420 grammes. The entire placenta is of a dull brownish color, and a distinct vascular trunk runs from the insertion of the cord, in the larger lobe, to the lesser lobe. On the maternal surface, the cotyledons are poorly marked, and here and there are seen a few minute areas of calcification. There is a large white infarct, 3 cm. in diameter, at one margin of the lesser lobe. The entire surface is crepitant, and in places small bubbles of gas escape on pressure. The fœtal surface shows numerous blebs and bullæ beneath the amnion, which contain gas. The larger placental vessels are distended with gas, which can be pressed into the smaller ramifications.

The umbilical cord is 36 cm. long, it is thin and flabby, and its vessels have considerable gas within them. Cover-slips made from the placental blood show large numbers of typical bacilli.

Microscopic examination of hardened portions of the placenta. The decidua presents for the most part a normal appearance. In the serotina the cells are often necrotic and have become converted into canalized fibrin, which in places is seen to be continuous with the canalized fibrin of typical infarct areas of the chorion. Infiltration with small round cells is noted to a slight degree. The decidua is poor in gland and vessel spaces, and when present they seem to have lost their epithelium and endothelium.

In that part of the chorion situated near the decidua serotina the villi present a typical appearance, they are closely packed together, rich in vessels, and their cellular elements take the stain well. Many small infarct areas are seen, particularly at the margins of the placenta, which present the usual structure. As we go deeper into the chorionic substance (*i. e.* toward the amniotic surface), we see that the villi do not lie in so close relationship with one another, but are widely separated.

The larger vessels of the chorion are seen to be plugged with bacilli, identical with those seen in cover-slip preparations mentioned in the macroscopic description. In some of these vessels the bacilli form a solid plug, completely filling the lumen, while in others a "gas cyst" has been formed, and the bacilli lie in a zone around its margin. As we go from the vessels into the chorionic substance we find a few bacilli; these do not lie in the villous capillaries, but in the stroma of the villi, thus it is seen that while the larger vessels are filled with organisms the capillaries are free from them. The "gas cysts" mentioned above are found to lie in the placental vessels and not in the intervillous spaces.

The tissue surrounding one of these foci of bacilli presents the following characteristics: Centrally we find the mass of bacilli, which may lie either closely packed together in a clump, completely filling the vessel lumen, or surrounding the margin of a "gas cyst" in a zone. Going outward we first find a zone of necrotic material, the remains of the vessel wall; this is entirely necrotic and shows no trace of nuclei. We next get a zone of necrotic villi, which take a pink stain with eosin, and show no cell structure. That portion of the villus which formerly represented its syncytial covering takes

the stain somewhat deeper than the stroma. Continuing outward, we next find a zone of partly necrotic villi, in which each villus has retained a portion of its cell structure.

Which portion of the villus (syncytium or stroma) first succumbs to the toxic action of the organism cannot be said, for in this zone of partly necrotic villi we sometimes see intact syncytium surrounding necrotic stroma, and in other villi the stroma is normal while the syncytium is represented by a narrow necrotic band. It is probable that the syncytium is the more resistant of the two, as more of the first variety of villi are seen.

There is apparently no reaction on the part of the tissue, for we find no infiltration with polynuclear leucocytes. In some of the sections we were able to demonstrate a few streptococci.

The epithelium and Wharton ian jelly of the cord are entirely necrotic, the vessel walls partly so, and show a few nuclei. The arteries are apparently free from bacilli; in the veins, however, they are very numerous, but show no tendency to occur in clumps.

Bacteriological report (by Dr. Lanier). Cultures from the placental blood show the *bacillus aerogenes capsulatus* mixed with staphylococci and streptococci. A pigeon was inoculated in the pectoral muscle with 1 cc. of bloody fluid from the placenta and died in about eight hours. At the autopsy there was extensive gaseous crepitation to be made out over the entire chest. On removing the skin, the subcutaneous tissue contained a number of gas bubbles. The muscles were extremely œdematus, of a reddish brown color, and disintegrated to the extent of being almost pulpified. Cultures made from this muscle grew out in the characteristic manner.

A rabbit inoculated in the ear vein with a 24-hour bouillon culture from the original placental blood was killed by a blow on the back of the neck and left for 24 hours. At the autopsy emphysematous crackling could be made out over the entire abdomen and chest, the subcutaneous tissues and all of the organs contained large quantities of gas, slips from which showed large numbers of encapsulated bacilli. Cultures from these organs gave the characteristic growth. The organism was also pathogenic for guinea-pigs.

Pathological diagnosis. Bacillus aerogenes capsulatus (Welch). Infection of placenta and fœtus.

In looking over the literature of the subject we have only been able to find a few cases which can be said to be similar to ours. The first of these cases was reported by Leduc[3] as early as 1597, he was called to see a woman who had been in labor for three days. After a very difficult extraction by means of the blunt hook, he says: "Immediately after the delivery of the body of the child and placenta, a violet-colored flame with a sulphurous odor escaped from the vulva, this flame shot out some distance from the external genitals of the patient, and its heat could be distinctly felt by the hands of the persons who held her." She died several days later. It is highly probable that this gas was generated by the bacillus aerogenes capsulatus, but we can hardly blame the author of that period for giving to his report of the case the somewhat fantastic title of "Le diable au corps."

Doleris[2] reported a case of a woman dead after labor, of a bacillar infection, this was before the work of Welch and Nuttall, but the clinical history is so characteristic of the form of infection we are discussing, a short quotation from his report will not be out of place.

"In 1883 I submitted to the Anatomical Society a case of delivery at term, complicated by a myoma, after which the patient died. The labor was exceedingly difficult and marked by successive intervention (forceps, cephalotripsy, etc.), and I was not able to deliver the fœtus. The patient succumbed to a septicæmia having its origin in an intense putrefaction of the uterine tissue, physometra, and putrid emphysema of the cellular tissue of the hypogastric region. I have studied the case carefully from a bacteriological point of view, and can affirm that it was a typical septicæmia caused by a bacillar organism and not by the streptococcus or staphylococcus."

In 1893 we find reports of two cases, one by Graham Stewart and Baldwin[3] and the other by P. Ernst.[4] As these cases were both worked up very carefully from a pathological and bacteriological standpoint, we give rather full abstracts.

Dr. Stewart[3] was called at 1 P. M. to see a woman who had been previously healthy and accustomed to hard work. He found her suffering with intense pain in the region of the uterus and ovaries. Miscarriage was suspected and denied. As the pain was somewhat relieved by morphia he diagnosed the case as acute metritis following abortion, and left the patient. He did not see her again until several hours later, during which time she became so much worse that another physician had to be called. At Dr. Stewart's third visit at 9.30 P. M., about eight hours after the onset, he found her emphysematous from "the top of her head to the soles of her feet." Shortly after this she died, and swelling of the body began, and increased so rapidly as to greatly alarm the undertaker when he was called in.

At the autopsy Dr. Baldwin reports "on making the usual longitudinal incision, gas bubbles were found in the subcutaneous tissue, which ignited with a characteristic explosion, and a small amount of gas in the peritoneal cavity. The abdominal veins are markedly distended with gas, and numerous bubbles found in all the internal organs. The uterus gave evidence of an abortion at about the third month." There is no record of microscopic examination of any of the organs.

Bacteriological examination of a specimen of blood showed the presence of the bacillus aerogenes capsulatus in large numbers.

It is very interesting to note that in this case production of gas took place to a very marked extent before death.

Ernst,[4] in doing an autopsy on a woman dead after operation for removal of a macerated fœtus, was struck by the appearance of foam on the cut surface of the liver, which on being allowed to stand increased so rapidly as to form a layer several centimeters thick.

Bacteriological examination proved this case to be one of infection with the bacillus aerogenes capsulatus. An abstract of his case is as follows.

Patient 26 years old, with anteflexion of the uterus in her first pregnancy. Entered the hospital suffering with pain in the right side of four weeks' duration. She had also lately

noticed a tumor in the lower abdomen. There being no definite signs of pregnancy, and the microscopic examination of scrapings from the uterus showing decidua, the cervix was dilated to admit manual exploration of the uterine cavity. Digital examination, however, revealing nothing, a vaginal tampon of iodoform gauze was introduced.

After this the patient became so much worse that the tampon had to be removed. A macerated fœtus was found and extracted, and the uterus repeatedly douched with antiseptic solutions; the patient, however, became steadily worse and died.

The autopsy showed fibrinous adhesions between abdominal wall and intestines; cloudy sanguineolent fluid in the pelvis. The musculature of the left ventricle is thickly beset with little miliary abscesses (?), in the centre of each of which is a small punctiform lumen. Fresh fibrinous exudate on the under border of the lungs. Spleen tumor with fibrinous coating and many necrotic areas appearing beneath its capsule. Cloudy swelling of both kidneys. In the liver, which is of a yellowish icteroid color, there are countless yellowish and whitish-gray foci, the size of a millet seed, in the centre of each of which there is a small lumen.

The uterus reaches to the umbilicus, it is 15 cm. long and its walls have an average thickness of 2 cm. The placental site occupies the anterior wall, and here the uterus is thicker (2½–3 cm). The entire uterine wall crackles on account of its containing numerous gas bubbles. The cervical portion of the uterus is 5 cm. long and is gangrenous. The uterine cavity presents a ragged meshed surface of a dirty gray color. A dirty, sanguineolent fluid flows from the venous plexus of the right broad ligament.

The uterus and ovaries are covered with a fibrinous exudate; in the right ovary is a corpus luteum of pregnancy, undergoing cystic degeneration. There is a clot in the right iliac vein which is riddled with gas bubbles.

Ernst was unfortunately prevented from making cultures at this time, and later the organism had died out.

In Ernst's article he goes very thoroughly into the histological changes produced in the internal organs. These changes are similar to those described by Welch and Nuttall, so they will not be mentioned in this report.

The changes mentioned in connection with the uterus are especially interesting to us owing to the fact that we were unable to get an autopsy in our case. Ernst mentions three different organisms: a large bacillus similar to those found in the internal organs, viz. the bacillus aerogenes capsulatus; a short thick bacillus which he considers a saprophyte; and cocci having a tendency to chain arrangement, however, as he says, not the typical chains as seen in streptococci. The short thick bacilli he finds only in the necrotic uterine mucosa, and they show no tendency to invade the deeper structure, while the cocci are found deep down in the uterine muscularis. The larger bacilli, which are found all through the internal organs, are very numerous in the uterine wall; they have formed "gas cysts," which give to the uterus the ragged, mesh-like appearance mentioned above.

In comparing these "gas cysts" with those found in the liver, Ernst says the principal difference is, that while in the liver the bacilli line the cyst in an unbroken zone, in the uterus the organisms are only found lining portions of the cyst wall, and the cyst has a much more irregular contour. This difference he attributes to the difference in structure of the two organs, that of the liver being more or less homogeneous, while the uterine wall is made up of numerous fibres running in different directions.

Thus, when a mass of bacilli begin to form gas in the liver, the resistance met with is equal in all directions and the bubble takes the form of a sphere. On the other hand, in the uterine wall, the resistance not being equal, the bubble when formed will have an irregular contour, and in places along its wall the bacilli will form a very thin zone or be entirely absent.

Ernst is in doubt as to whether the infection took place along the veins or lymphatics, for in his specimens both contained large numbers of bacilli.

In 1895, Krönig,[6] at the Sixth Congress of the Deutsche Gesellschaft für Gynäkologie, reports two cases of puerperal endometritis probably due to the bacillus aerogenes capsulatus.

During the last year Schnell[7] reports a case which, although he does not consider it an infection, has a clinical history so similar to our case that a short abstract will not be out of place.

Primipara, æt. 31. 24 hours after rupture of the membranes was seen by a midwife from the Polyclinic, who found prolapse of an arm. Version was tried, but failed, on account of contraction of the uterus; the child died during this attempt. She was brought to the hospital with marked *tympania uteri* and suffering intense pain; 46 hours after the onset of labor she was delivered by a difficult version. Immediately after labor she did well, but later had quite a profuse hemorrhage, for which the uterus was tamponed with iodoform gauze. She then became steadily worse, and died three and a half hours after delivery.

At the autopsy 18 hours after death, in winter, nothing abnormal was found but gas in the large veins of heart, pericardial vessels, lungs, aorta, liver and spleen.

There is no record of any bacteriological examination, and Schnell considers the death due to air having gained entrance into the uterine sinuses during the successive manipulations.

On looking over and comparing these cases with our own, the following points are of interest: In every case the infection was very rapid and fatal. In all of the cases except that of Graham Stewart and Baldwin, there was for some time a dead fœtus in the uterus, and the organism found an excellent medium for development in its dead tissues and in the placenta, and probably did not gain entrance to the general circulation until after death of the patient. In the case of Graham Stewart and Baldwin, on the other hand, there was nothing in the uterine cavity; it only "gave evidence of a recent abortion." Here it is probable that the infection was more in the nature of a true septicæmia, and the bacilli gained direct entrance to the vessels and lymphatics at the abraded placental site. As this was the only case in which gas was detected in the subcutaneous tissues before death, the above theory is not out of the line of probability.

In Schnell's case we feel sure that had a bacteriological examination been made, the organism would have been found.

There are in our case two questions which deserve consideration, in the first place, the origin of the infection, and in the second, the action of the organism after it had entered the body.

The first question, in the absence of an accurate subjective history, cannot be answered with certainty. Suffice it to say that as the patient had been in labor two days before we were called in, and during that time a midwife had been in constant attendance, it is more than probable that the organisms were introduced by her during her examinations.

In regard to the action of the organism after it entered the body, *i. e.* whether the case was one of sapræmia or septicæmia, we can say as follows: Welch and Nuttall[1] have proved by inoculation into rabbits, that when the organism is introduced into the general circulation, death of the animal does not follow, but if shortly after the inoculation the rabbit be killed, the bacillus develops very rapidly, with abundant formation of gas in the various organs.

In their experiments on rabbits, in only one case did the inoculation prove fatal. This occurred in a pregnant rabbit in which two of the embryos were dead and macerated at the time of the experiment. Here the bacilli gained foothold and developed.

From the above we can then argue that the organisms were introduced by the midwife early in labor. She failed to recognize the obstruction offered by the deformed pelvis, and allowed the case to go on. The child, after becoming firmly impacted in the pelvis, died, and the bacilli, which had before undergone little development, now having the most favorable culture medium for their growth, *i. e.* dead tissue deficient in oxygen, increased to an enormous extent and partly filled the uterine cavity with gas.

Thus, by the time we were called in to see the patient she was not only weakened by the prolonged labor, but also profoundly poisoned by the toxines produced in the uterine cavity.

Why did not her general condition improve after removal of the dead fœtus and placenta? To this it may be replied that the fœtus being so long under pressure, there was considerable sloughing and necrosis of the cervical and vaginal tissues; thus there was still a large amount of favorable media which we could not remove.

One other point deserves consideration. Did the infection of the fœtus and placenta take place before or after fœtal death?

Again referring to the experiments of Welch and Nuttall, the fact of our finding the bacilli and "gas cysts" only in the lumina of the placental vessels is a strong point in favor of the theory that, just as in the mother, the organisms entered the blood before death, but did not undergo rapid development until the fœtus died.

From the above I think we can consider our case as one of septic intoxication rather than a septic infection.

Since writing the above report we have been told by Dr. N. G. Kierle, City Autopsy Physician, that it is not a rare thing to find conditions similar to these, viz. production of gas in the subcutaneous tissues and internal organs, in cases dead after criminal abortion.

References.

1. Welch and Nuttall: A gas-producing bacillus (bacillus aerogenes capsulatus) capable of rapid development in the blood-vessels after death. Bulletin of the Johns Hopkins Hospital, July–August, 1892.

2. Welch and Flexner: Observations concerning the bacillus aerogenes capsulatus. Journal of Experimental Medicine, Vol. I, No. 1.

3. Graham Stewart and Baldwin: Bacillus aerogenes capsulatus — Case. Columbus Medical Journal, August, 1893.

4. P. Ernst: Ueber einen gasbilden Anaeroben im menschlichen Körper, und seine Beziehung zur "Shaumleber." Virchow Archiv, Band 133, Heft II.

5. Leduc: Le diable au corps. Witkowski's Accoucheurs et Sage-femmes célèbres, p. 184.

6. Krönig: Discussion on Endometritis. VI Congress Deutscher Gesellschaft für Gynäkologie.

7. Schnell: Ueber einem Fall von gasblasen im Blut einer nach tympania uteri gestorben Puerpera. Monatsschrift für Geburtshülfe und Gynäkologie, IV, 199–203, 1896.

8. Doleris: Inflammation puerperal. Arch. d'Obst. et de Gyn., Vol. IX, pp. 97–122, and pp. 142–161.

Discussion.

Dr. Flexner.—I was enabled, through the courtesy of Dr. Dobbin, to see, during life, the patient whose case he reports. At my first visit, some 6 or 8 hours previous to her death, I endeavored to ascertain if there was any evidence whatever of an ante-mortem development of gas within the tissues or vessels in parts remote from the genitalia. No such development was discovered. The next time I saw the case was several hours (6 or 7) after death. At this time the appearances presented were those of extensive gas formation everywhere in the soft tissues and serous cavities. From the nose and mouth frothy bloody serum exuded, which on cover-slip preparations showed many characteristic bacilli. From the account given by the family it was clear that rapid multiplication of the organisms had begun almost at once after death, thus indicating a general distribution of the bacilli during life. I should prefer to consider that other causes than merely the presence of oxygen in the circulating blood are to be taken into account in explaining the absence of any appreciable development of gas, and thus presumably of bacilli, in the body during life. I apprehend that still other factors play a part, and these probably are of the same nature as are reckoned with in other and more usual forms of infection with bacteria. This case is, I believe, the first one which has been encountered in the Hospital in which the invasion followed parturition. It is in itself a confirmation of the prediction made originally by Welch and Nuttall, namely, that many of the cases of supposed entrance of air into the uterine sinuses would be found to be due to infection with a gas-producing micro-organism. The prediction had, however, been abundantly confirmed before by Ernst and Graham, as related in Dr. Dobbin's paper.

PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS HOSPITAL MEDICAL SOCIETY.

Meeting of October 19, 1896.

Dr. THAYER in the chair.

The Surgical Significance of Gall Stones.—DR. F. LANGE.

Every physician who has practised for a long time knows that there are cases in which through a number of years attacks of gall stones will recur and eventually the patient will be entirely cured. I have, for instance, for a number of years watched a colleague of mine, a physician, who beyond any doubt has had gall stones, and indeed I have felt the gall stones in his dilated gall bladder. He had all the symptoms of cholangioitis, with frequent chills and high fever, through months and years. He, however, did not like to undergo surgical treatment, and he is now in flourishing health.

I have seen an old lady with a gall bladder packed with gall stones, who was not operated upon, who never had any severe attacks that had been taken for gall stones, but in whom apparently the gall stones have disappeared without any serious symptoms. Pathologists know that the presence of gall stones, especially in elderly persons, at autopsies, is of very frequent occurrence. On the other hand, the disease may call for very prompt and active surgical treatment, and it depends entirely upon the pathological changes—the consequences of the presence of gall stones or their accompanying changes in the gall system—whether the case will present features that call for surgical treatment.

I propose to accompany a gall stone from its usual place of residence through the gall passages, and on this journey I shall mark out some clinical features which we will occasionally be brought in contact with. As long as the gall stones remain quiet in the gall bladder they will hardly ever give rise to any symptoms, but as soon as they become dislodged and commence to travel into the cystic duct or the common duct, then the typical symptoms will commence. They may sometimes grow to a large size and cause very few symptoms. I show you here a specimen of gall stones that came from a lady who for a number of years had been suffering from "dyspepsia," but not until an acute attack of peritonitis had set in, in consequence of impaction of this very large gall stone in the cystic duct, was she ever supposed to be suffering from gall stones. As you see, the gall stones are almost the size of a hen's egg. The gall bladder was full of pus, and peritonitis had commenced. Acute impaction of the gall stone in the cystic duct, in my experience, happens not unfrequently with solitary stones of larger size. Since the first of last October I have had six such cases of acute cholecystitis with septic processes on the surface of the mucous membrane of the gall bladder, or through the entire thickness of the gall bladder, which brought the patient into more or less danger of life. In one of these cases, in which I had operated three years previously, I had the opportunity of observing the changes that had taken place three years after operation. The patient was left with a pretty extensive scar and a hernia formed. She got an acute strangulation of a small omental hernia which necessitated incision. It was in the immediate vicinity of the gall bladder, and the gall bladder on that occasion was opened also. I found, to my surprise, that the gall bladder had assumed an hour-glass shape, through the loss of substance which had occurred in the expulsion of necrotic tissue in the first attack, and there existed two compartments communicating with each other by means of a very narrow canal. In both of these compartments was a considerable amount of mucous material, but no stones. The patient had suffered at the time from a certain degree of catarrh of the gall duct.

I have here a stone of not much smaller size, made up of cholesterine with lime outside. The patient had an acute attack of cholecystitis, and this stone was found impacted in the cystic duct.

This third stone, of smaller size, is from a case in which the destruction of the gall bladder was quite extensive. Eventually the patient made a good recovery.

Solitary stones are not always the cause of this affection. On the contrary, sometimes quite the opposite condition will be found. You will see here in this bottle a large quantity of gravel-like material, coming from the gall bladder of a gentleman who for several years had been complaining of pain about the region of the liver. In the second bottle you will see an amount of tissue coming from the disintegrated mucous membrane, and if you will take a closer look at these pieces you will see that a large number of these small black granules are imbedded in these shreds of membrane. The gall bladder was felt through the patient's lifetime as a very hard, resistant, stony body, and when I tried to remove it it broke off in the region of the cystic duct, and I was able to feel below a hard resisting mass. The patient was in such a weak condition that I did not try to get any further. After about six months she died from a progressing carcinomatous affection of the cystic duct. The cancer of the cystic duct pressed the common duct and led to jaundice and marasmus.

If the gall stone passes beyond the cystic duct into the common duct and is arrested there, of course the symptoms of stagnation of the bile will make their appearance. Very frequently, however, associated with this there is inflammation of the gall system, such as cholangioitis or pericholangioitis or even liver abscess. These cases often necessitate very difficult tasks, and I must say that some of them upon which I have operated are among the most difficult operations I have done. They are, however, very successful operations, and operations by which the life of the patient is saved. In one of them I was able to push the stone back through the dilated cystic duct into the gall bladder, and then did what may be called an ideal cholecystotomy, that is to say, I sewed the gall bladder up again and closed the abdomen. That was ten or eleven years ago; of course I would neither do it again nor would I recommend it. It is evident that through the mechanical injury of the operation and through the inflammatory condition which is present, a certain amount of swelling is kept up for a time, and then the duct will not immediately become patulous after

the sewing of the wound, and so it was in this case. The sewed-up gall bladder was put upon a tension and burst, and the patient died from sepsis.

I have here a small cholesterine stone which led to a bad condition of the patient. A lady, about forty years of age, the wife of a physician, who was very carefully observed, had had one attack of gall stones about four or five months previously to the time I saw her. She was taken with chills, high fever and deep jaundice. I found her, about nine or ten days after the beginning of this attack, in a very low condition, so bad that the only indication at that time was to open the gall bladder, drain it and do away with the stagnation of the bile. About three weeks later this stone was removed from the common duct, and although the patient for a number of months after this presented some febrile symptoms, with occasionally slight chills, she eventually has entirely recovered.

Here is another stone, which has been removed from the ductus choledochus, and one of exceptional size for a choledochus stone.

I have one specimen in which I was obliged, apart from opening the gall bladder, to incise the cystic duct at two different places, and also the common duct. That was a case in which the gall stones were very small, not larger than peas, and which were caught behind the protruding folds and diverticula of the cystic duct, so that they could not be shifted. I sewed up the common duct as well as the cystic duct, and drained the gall bladder. This patient made a good recovery.

The principal point of resistance for gall stones is the region of the papilla in the wall of the duodenum. In a good many instances it has been found that large stones there destroy the surrounding parts and form fistulous communications with the duodenum. I have had the opportunity of operating in one such case, in which I thought I had to do with a malignant affection of the duodenum. The patient was sent to me as a case of cancer of the pylorus. It could be made out easily that the pylorus must be free, because vomited matters contained a large amount of bile. By palpation nothing certain could be made out, and in operating, I cut down through a great many adhesions and a good deal of scar tissue and found the duodenum obstructed by a hard mass, apparently of scar formation. I separated that, but in going further I met with a hard firm body which was apparently adherent to the vertebral column. I stopped the operation. The patient was very much run down. Believing that the duodenum was not sufficiently patulous I made a gastro-enterostomy. The patient rallied from the operation fairly well, but fell into a state of collapse five or six hours later and died. In making the autopsy I found, to my very great surprise, that this large mass behind the duodenum was not a malignant affection, but a large mass of gall stones, partly lodged behind the wall of the duodenum and partly within the wall of the duodenum, with a number of fistulous openings into the duodenum. The constrictions from scars were such that the contents of the duodenum could not easily pass beyond this point, and the symptoms of obstruction of the intestinal tract were the consequence.

I show you here a stone which has been excised from the small intestine. It is a very beautiful specimen of a pure cholesterine stone. Why a stone of this size cannot pass, while a Murphy's button will pass easily, I cannot tell. The patient in this case was an old lady. The operation was done very smoothly and I had good hopes for recovery, but she died in eight hours after the operation, from collapse.

These are the regular ways of the gall stones; but we know that from the gall bladder or from any other point in the gall system, through destruction of the surrounding tissues, they may find an outlet into a neighboring viscus or through the abdominal wall. Cases of this kind have at all times been very interesting and full of medical interest. I show you here specimens from one of my first cases of this kind, which occurred about seventeen years ago. It was a case of abscess of the abdominal wall, at the bottom of which I found gall stones. They were still lodged in the gall bladder. The patient would not submit to further surgical treatment and still has a fistula which discharges bile.

I have here some specimens, some 300 in number, from a case of acute cholecystitis. And here are specimens from another case of cholecystitis, in which I might call your attention to a point of interest, namely, on the cross-cut you see three small gall stones matted together by a binding mass, so that a single stone is simulated. Gall stones in the first stages of their development are very soft masses, and, owing to the beautiful and careful researches of Naunyn, we know that there is an organic substructure for their development.

A very serious complication that has led to fatal issue in several of my cases of advanced cholemia is an inclination of the patients to bleeding. Three of my cases I have lost from secondary hemorrhages. Two of these cases were complicated with malignant disease of the pancreas; at least I think they were cases of cancer of the pancreas. Autopsies were not made, and I can only say that I felt in the region of the head of the pancreas a resistant hard mass which I took for a malignant affection. Lately, Professor Riedel, a surgeon who has perhaps worked more in gall surgery than any other living man, has published cases in which he is inclined to assume that occasionally these apparent malignant thickenings of the pancreas may be but inflammatory thickenings, and he mentions one case which seems to be beyond any doubt. In an elderly gentleman, in whom the operation was abandoned, assuming that this was a case of cancer of the pancreas, the stones merely were removed. A fatal issue was awaited, but the patient lived for years and years and became healthy and strong, and there could be no question about his not having malignant disease. He assumes that through the presence of stones an irritation is kept up in the pancreas, and that this irritation, after the removal of the stone, may cease and the condition improve.

I have lost one of these cases by the separation of the Murphy's button after establishing cholecystenterostomy. The patient had had up to that time small hemorrhages from the inside of the gall bladder. I had purposely kept the gall bladder open, stitched to the abdominal wall, because I had the impression that this probably would be a case of bleeding, and in order to tampon and make counter pressure I kept the gall bladder open, and was able up to the tenth or eleventh day to check the bleeding, but after the separation of Murphy's

button the patient had a profuse hemorrhage into the large intestine and succumbed to anæmia.

In another case there was no proof of any malignant infection. It was a young person with impaction of gall stones in the common duct. The operation went off very well, but on the fifth or sixth day she had a secondary intra-peritoneal hemorrhage which was, I am sorry to say, detected too late. It is a fact that has been observed repeatedly, that in some cases there is an inclination to hemorrhage a certain number of days after operation. These patients who are apt to bleed from cholæmia, show a great tendency to bleed while one is incising the abdominal wall. They are, as a rule, much run down in health.

I will show you here an exceptionally beautiful specimen of gall stones from a case of sarcoma of the gall bladder,—a sarcoma which was not detected at the time of operation. I thought it was a case of stenosis from impaction of a stone in the apex of the cystic duct. To be sure, there was a suspiciously rough surface at the point of this stone. After a number of months a tumor mass sprung up from the gall bladder. Evidently this stone had been lodged with its point on the irregular surface of the tumor, and I think the roughness of the stone is somewhat indicative of its being in contact with an irregular surface and not with the smooth surface of the gall bladder.

As far as the surgical technique of this operation is concerned: To begin with the abdominal incision, I must say that I have entirely abandoned the longitudinal incision; but, according to the proposition of Corvisart and the experience of other surgeons, I do exclusively the operation parallel to the border of the ribs. Correspondingly to the thickness of the abdominal wall, this incision occasionally must be a very large one. A very great difficulty must occasionally be met with through the smallness of the liver. It is an entirely mistaken idea that in consequence of stagnation of bile the liver must be enlarged. On the contrary, I have had the experience that in occlusion of the common duct of long duration, probably through the influence of pressure, an atrophic condition of the liver tissue will take place, and occasionally the liver will be concealed high up behind the ribs, and this may cause a great deal of difficulty in consequence of the high and deep position of the field of operation. I have taken, in eight of these cases, to excising the adjacent cartilages of the ribs, and I should like to commend that. It facilitates the operation very much. On the other hand, of course the enlargement of the liver may be a great hindrance, and, especially in fat and plump persons of short build, it may be quite difficult to get down to the common duct. In one of the cases I was unable, in spite of excision of the ribs, to get effectually down to the point of the trouble, and not until I had pulled the liver as far as I could out of the incision, almost one-third of the liver substance being brought outside of the abdominal wall, was I able to easily get to the cystic and common ducts. It was the case in which I was obliged to open the cystic duct at two different points and, besides that, the common duct at the usual place about the middle of its course. In the cases of acute inflammation it is of course necessary, wherever one has reason to presume that the neighborhood of the gall bladder is infected, to keep everything as much as possible open and give entirely free outlet for the peritoneal secretions. At the same time I always drain the gall bladder with a long tube with the view of having a siphon action upon the secreted gall, and this will act very effectually. Of course occasionally a blockage will take place through mucus or blood clot, and then it will be necessary to change the tube or make a cautious washing through it. I should advise against the injection of any irritating antiseptic solution into the gall bladder. I have observed that occasionally this fluid will cause distressing symptoms, attacks of colic, probably because it may get into the common duct and the hepatic duct and irritate. I only use saline solution or boiled water. I have always found that the bile itself was an antiseptic agent. After a short time the amount of purulent secretion is insignificant, and the healing of the abdominal wounds, although constantly in contact with the bile, is mostly uneventful and without any untoward symptoms.

About stones in the liver substance I have no practical experience. I have once found a stone in the liver after it had perforated the walls of the gall bladder, but I have not had any experience with stones in the liver that have formed in the gall system above the hepatic duct. It is a fact that these stones will occur, and that occasionally quite extensive casts of stone formation will take place in the gall system.

I will not mention comparatively frequent uncomplicated cases, where we have to deal with dilated gall bladders and stones and where the surgical treatment is comparatively simple.

The operation in two sittings I never do. I find that it is entirely safe to operate in one sitting. I have always aspirated as much as possible the contents of the gall bladder through a thick aspirating needle, protecting as cautiously as possible the surroundings to guard against infection.

DISCUSSION.

DR. HALSTED. Dr. Lange's talk has been instructive to all of us. At one of our meetings, not very long ago, I described three consecutive choledochotomies or incisions into the common duct for the removal of gall stones, all of which were successful. Two of these cases I exhibited on that occasion. What Dr. Lange says about the enlargement of the head of the pancreas interests me, because on several occasions I too have noticed in gall-stone cases that the pancreas was hard. On the first occasion, some four or more years ago, I was misled and thought for a time that the patient might have cancer of the pancreas. Since then I have several times found an indurated pancreas in these cases and have spoken of it at the operating table. The induration of the head of the pancreas was especially noticeable in one case. This case was one of great interest irrespective of this hardening. A very large stone occupied and had almost ulcerated through the common duct. There was, however, no local peritonitis. A little scratch and a little pressure on the wall of the common duct were enough to extrude the stone. The stone being extracted, we found quite a large hole in the common duct and duodenum just where they came together. This made a plastic operation necessary; and a very difficult one it proved to be

because we had to do such delicate work at such a very great depth. Even if the parts to be stitched could have been brought to the surface it would have been no easy task to accomplish the repair of the hole. The patient made an excellent recovery. She has gained nearly *one hundred pounds* and has no symptoms of stricture of the ductus choledoctinus. Of the other cases of stone in the common duct, one is interesting because complete obstruction was caused by a very small stone in the diverticulum of Vater, or near it. This stone, not more than 3 mm. in diameter, could not be pushed in either direction. After considerable manipulation we cut down upon it and removed it. It will undoubtedly strike you as very strange that we could not manage to extrude so small a stone. We might possibly have succeeded with the exercise of some violence; but the choledochotomy recommended itself to me as the safer procedure. We might, with a hypodermic syringe, inject oil into the duct and so perhaps make smooth the folds of mucous membrane and dislodge the stone by subsequent manipulation.

Regarding deaths from hemorrhage: Dr. Lange speaks of the so-called parenchymatous hemorrhage in these highly jaundiced patients. We have had one death from this cause. Our first common duct case died on the tenth day from hemorrhage. There were absolutely no symptoms of peritonitis. The patient made a very good recovery from the operation, notwithstanding the fact that he was so weak that we hesitated to operate upon him.

In 1880 or 1881 I performed my first gall-stone operation. At that time only one or two operations for gall-stones had been performed in this country. The case is one of considerable interest. Seven stones were removed from the bladder, which was distended with pus and adherent to the abdominal wall. The common duct could not with safety have been examined even if it had occurred to me to do so. The patient lived for about two years after the operation. At no time after the operation was she entirely free from jaundice, nor was she at any time either before or after the operation profoundly jaundiced. The stools were usually acholic, but occasionally showed unmistakable signs of bile. The sinus which led to the gall-bladder was not allowed to close until a few weeks before the death of the patient. Death was precipitated by a slight local peritonitis. At the autopsy all signs of the sinus which had led to the gall-bladder had disappeared. Nothing but a few connective tissue threads remained to indicate where the sinus had probably been. The gall-bladder was reduced to nothing but a little knob of connective tissue. The liver was perhaps less than one-half its original size. The common duct and one hepatic duct were dilated sufficiently to permit a calculus as large as a plover's egg to glide easily up and down from the duodenum to the interior of the liver. The calculus was somewhat egg-shaped, and could almost be pushed into the duodenum through the opening which partly by ulceration, partly by dilation, it had made for itself. It became engaged at this ring of cicatricial tissue a little beyond its middle when allowed to fall naturally, point downwards, in the common duct, from the liver to the duodenum. The wall of the common duct was very thick. The stone had evidently freely traversed the entire length of this dilated canal, from the very centre of the liver to, and almost into the intestine. At the autopsy which was made by a very distinguished pathologist the calculus was not found until the liver had been literally cut to pieces, so deeply was the stone imbedded in this organ. The occasional presence of bile in the stools and the absence of profound jaundice were thus readily explained by the findings at the autopsy. When the calculus was in the dilated branch of the hepatic duct, bile could escape through its other branch and appear in the feces. When the calculus plugged tightly the orifice into the duodenum the jaundice increased. That it should never have been profound could be explained by the fact that the stone traveled freely everywhere in the duct; a little bile could probably escape except when the stone was engaged at the duodenal end of the duct.

Will Dr. Lange kindly tell us if he always employs drainage of some kind in suture of the ductus choledochus; and if so, how long does he permit his drain to remain? Does he not think that the drainage material, gauze, etc., should be left undisturbed until the rest of the peritoneal cavity is well walled off and protected from reinfection? Quite recently one of Germany's best surgeons removed the gauze in one of these cases on the third day. Peritonitis supervened promptly and the patient died. I have noted that this surgeon and several well-known English surgeons state that no harm is done by leakage from the duct after suture of it, provided one drains down to the sutures. I think that one should, if possible, so suture the duct that its contents cannot escape. Fluid can convey solid and infected particles to places in the general peritoneal cavity which are not protected by the gauze. Furthermore, I have the idea that adhesions take place more firmly about dry gauze than about gauze which is constantly bathed in fluids.

For this reason, if we have sutured a gall-bladder to the parietal peritoneum, or if we have packed gauze anywhere about this gall-bladder which we have opened and propose to drain, we endeavor to prevent leakage for the next three or four days by passing a purse-string suture all along the edge of our opening into the gall-bladder, and drawing it up when we have introduced our gauze or drainage tube into the gall-bladder.

We explore all the ducts and open and evacuate the gall-bladder in one act. Riedel's advice to perform the operation in two acts, opening and evacuating the gall-bladder at the second act, is bad. We have more than once, while removing biliary calculi, remarked that it would have been impossible to extract them without the aid of fingers in the abdomen. Bimanual manipulation is of course impracticable when the bladder is adherent, all around, to the abdominal wall, as it is at the time of the second act in the operation in two acts.

Dr. Lange's suggestion to cut through one or two ribs to facilitate matters where the liver is very small and the gall-bladder so high as to be otherwise almost inaccessible, is undoubtedly an excellent one.*

*Jan. 2nd, 1897. I have recently had occasion to resort to this procedure while removing a tuberculous kidney which was adherent to the diaphragm. After dividing two ribs I could proceed quite easily with a dissection which otherwise might not have been feasible. When the liver is very large this method of gaining room is indicated quite as much a when the liver is small.

DR. LANGE.—I think that in some of my cases I had reason to assume that there was some valve action, especially in one case of stone in the common duct of small size. The patient had had an attack of gall stone some three months before this attack, with jaundice lasting several weeks, most likely because of obstruction from this stone, which was solitary, as judged by its very round, regular shape. Probably after a time this stone shifted back and later on again caused obstruction together with an infection. This we saw not uncommonly in cases of obstruction of the common duct. Even if the stones are ball-like or rounded, at intervals a certain amount of bile will get into the gut. It is almost characteristic that if, in prolonged jaundice, at intervals the condition of the fæces and the urine points to the passage of a certain amount of bile through the duct, we have probably to deal with obstruction from stone.

Regarding the drainage of the common duct, I have tried everything: I have left the duct entirely open, and I have sewed it up with the insertion of a small drainage tube into the lumen of the duct, and I have sewed it up entirely. In cases where the wall of the common duct is healthy, I think it is preferable, if it can be done, to sew up the duct entirely. If there is reason to assume that there is obstruction beyond the stone, I should prefer to drain, and I do this in such a way that I insert a small drainage tube from the wound in the common duct and pack gauze around it. This gauze (iodoform gauze boiled in glycerine) I leave in for five or six days. The tube I leave for a sufficiently long time to secure the proper outlet of any secretion as long as it exists. In draining the gall bladder I pack the gauze around the tube and leave the opening comparatively large, for the reason that, at least in inflammatory cases, the surface of the gall bladder is such that it will necessitate some thorough cleansing and local treatment for a limited time. There will be clots of blood or shreds of tissue or inspissated mucus which might easily obstruct. I mostly remove this tampon in two or three days and give the inside of the gall bladder an additional thorough washing and cleansing.

Besides this we have either sewed the peritoneum to the surface of the gall bladder where there is no infection outside of it, or we have packed around the circumference of the gall bladder in such a way that even if some of the contents of the bladder escape it is not likely to cause a spreading inflammation.

Dr. Halsted has spoken of the necessity of having a proper needle; I have a proper needle.

In one case I dissected the peritoneum from the walls of the abdomen. The case was that of an old gentleman, a physician, who had been suffering for years from attacks which were supposed to be stomach attacks, and there was a suspicion of his having chronic ulcer of the stomach and malignant induration. I opened in the middle line and found nothing, but on palpating the gall bladder I found it full of small stones. The liver was small and the gall bladder very high up. I was obliged to loosen the peritoneum. This gentleman had for five or six days incessant vomiting and the gall bladder was torn away from the peritoneum. He still has his gall stones in his gall bladder, but for a number of years he did not have any attacks. Lately, I understand, he has had them again.

I have also done the sewing of the omentum against the gall bladder in the way that Lauenstein proposes, and have been quite satisfied with it.

Finally, I should like to say that I have regarded the invitation to come here as a very flattering one. I have come with the greatest imaginable pleasure, and for the reason, apart from the friendly relations which I have here, that I have the consciousness of being at a place where science is promoted and sought for in a devoted and unselfish manner. I regard this institution as the pride of this country, and I hope that it will not be the only one,—that others will follow, and that this country will be eager to base medicine on the sound basis of scientific conception.

CORRESPONDENCE.

A CASE OF PNEUMO-CARDIAL RUPTURE.

To the Editor of the JOHNS HOPKINS HOSPITAL BULLETIN.

Dear Sir:—I send herewith a report of a case of pneumo-cardial rupture which has come under my observation, which you may deem of sufficient interest to publish.

On March 26, 1896, a negro man, age 23, was brought to our hospital in a condition of collapse, with the history that a bank of sand or earth had fallen on him and a fellow-workman, the latter dying in a few hours afterwards from internal injuries. The patient's breathing was shallow, jerky, rapid and very painful, and every few minutes he had a hard spasm of coughing, which distressed him greatly and during which he expectorated a good deal of bright red blood. Morphin to relieve his pain and coughing, and strychnin were given him freely. On the second day he complained of intense pain in the cardiac region, and the stethoscope revealed a very interesting, if not an entirely unique state of affairs. This was a loud double friction sound with a bubbling and splashing sound, which could be distinctly heard with the stethoscope two inches from the chest wall. The diagnosis was a rupture between the pericardium and the lung. On the third day (losing sight of the fact that air from the lung is usually sterile) I supposed that he had a septic pericarditis due to the effusion of blood and air from the lung. An examination of the blood, however, showed no leucocytosis, which proved conclusively that the pericarditis was not septic. This examination was repeated daily for three days, each time with the same result; and as at no time had he much fever, and as the pulse and respiration during this three days gradually improved, a favorable prognosis was given, which was justified later by his complete recovery. The loud splashing and bubbling disappeared by the fourth day. The rough friction sound persisted for three weeks, and after that the heart sounds were in every respect normal.

GEORGE S. BROWN, M. D.

2220 FIRST AVENUE, BIRMINGHAM, ALA.

Dec. 16, 1896.

PUBLICATIONS OF THE JOHNS HOPKINS HOSPITAL.

THE JOHNS HOPKINS HOSPITAL REPORTS.

VOLUME I. 423 pages, 99 plates.

Report in Pathology.

The Vessels and Walls of the Dog's Stomach; A Study of the Intestinal Contraction; Healing of Intestinal Sutures; Reversal of the Intestine; The Contraction of the Vena Portae and its Influence upon the Circulation. By F. P. MALL, M. D.
A Contribution to the Pathology of the Gelatinous Type of Cerebellar Sclerosis (Atrophy). By HENRY J. BERKLEY, M. D.
Reticulated Tissue and its Relation to the Connective Tissue Fibrils. By F. P. MALL, M. D.

Report in Dermatology.

Two Cases of Protozoan (Coccidioidal) Infection of the Skin and other Organs. By T. C. GILCHRIST, M. D., and EMMET RIXFORD, M. D.
A Case of Blastomycetic Dermatitis in Man; Comparisons of the Two Varieties of Protozoa, and the Blastomyces found in the preceding Cases, with the so-called Parasites found in Various Lesions of the Skin, etc.; Two Cases of Molluscum Fibrosum; The Pathology of a Case of Dermatitis Herpetiformis (Duhring). By T. C. GILCHRIST, M. D.

Report in Pathology.

An Experimental Study of the Thyroid Gland of Dogs, with especial consideration of Hypertrophy of this Gland. By W. S. HALSTED, M. D.

VOLUME II. 570 pages, with 28 plates and figures.

Report in Medicine.

On Fever of Hepatic Origin, particularly the Intermittent Pyrexia associated with Gallstones. By WILLIAM OSLER, M. D.
Some Remarks on Anomalies of the Uvula. By JOHN N. MACKENZIE, M. D.
On Pyrodin. By H. A. LAFLEUR, M. D.
Cases of Post-febrile Insanity. By WILLIAM OSLER, M. D.
Acute Tuberculosis in an Infant of Four Months. By HARRY TOULMIN, M. D.
Rare Forms of Cardiac Thrombi. By WILLIAM OSLER, M. D.
Notes on Endocarditis in Phthisis. By WILLIAM OSLER, M. D.

Report in Medicine.

Tubercular Peritonitis. By WILLIAM OSLER, M. D.
A Case of Raynaud's Disease. By H. M. THOMAS, M. D.
Acute Nephritis in Typhoid Fever. By WILLIAM OSLER, M. D.

Report in Gynecology.

The Gynecological Operating Room. By HOWARD A. KELLY, M. D.
The Laparotomies performed from October 16, 1889, to March 3, 1890. By HOWARD A. KELLY, M. D., and HUNTER ROBB, M. D.
The Report of the Autopsies in Two Cases Dying in the Gynecological Wards without Operation; Composite Temperature and Pulse Charts of Forty Cases of Abdominal Section. By HOWARD A. KELLY, M. D.
The Management of the Drainage Tube in Abdominal Section. By HUNTER ROBB, M. D.
The Gonococcus in Pyosalpinx; Tuberculosis of the Fallopian Tubes and Peritoneum; Ovarian Tumor; General Gynecological Operations from October 15, 1889, to March 4, 1890. By HOWARD A. KELLY, M. D.
Report of the Urinary Examination of Ninety-one Gynecological Cases. By HOWARD A. KELLY, M. D., and ALBERT A. GHRISKEY, M. D.
Ligature of the Trunks of the Uterine and Ovarian Arteries as a Means of Checking Hemorrhage from the Uterus, etc. By HOWARD A. KELLY, M. D.
Carcinoma of the Cervix Uteri in the Negress. By J. W. WILLIAMS, M. D.
Elephantiasis of the Clitoris. By HOWARD A. KELLY, M. D.
Myxo-Sarcoma of the Clitoris. By HUNTER ROBB, M. D.
Kolpo-Ureterotomy. Incision of the Ureter through the Vagina, for the treatment of Ureteral Stricture; Record of Deaths following Gynecological Operations. By HOWARD A. KELLY, M. D.

Report in Surgery, I.

The Treatment of Wounds with Especial Reference to the Value of the Blood Clot in the Management of Dead Spaces. By W. S. HALSTED, M. D.

Report in Neurology, I.

A Case of Chorea Insaniens. By HENRY J. BERKLEY, M. D.
Acute Angio-Neurotic Oedema. By CHARLES E. SIMON, M. D.
Haematomyelia. By AUGUST HOCH, M. D.
A Case of Cerebro-Spinal Syphilis, with an unusual Lesion in the Spinal Cord. By HENRY M. THOMAS, M. D.

Report in Pathology, I.

Amœbic Dysentery. By WILLIAM T. COUNCILMAN, M. D., and HENRI A. LAFLEUR, M. D.

VOLUME III. 766 pages, with 69 plates and figures.

Report in Pathology.

Papillomatous Tumors of the Ovary. By J. WHITRIDGE WILLIAMS, M. D.
Tuberculosis of the Female Generative Organs. By J. WHITRIDGE WILLIAMS, M. D.

Report in Pathology.

Multiple Lympho-Sarcomata, with a report of Two Cases. By SIMON FLEXNER, M. D.
The Cerebellar Cortex of the Dog. By HENRY J. BERKLEY, M. D.
A Case of Chronic Nephritis in a Cow. By W. T. COUNCILMAN, M. D.
Bacteria in their Relation to Vegetable Tissue. By H. L. RUSSELL, PH. D.
Heart Hypertrophy. By WM. T. HOWARD, JR., M. D.

Report in Gynecology.

The Gynecological Operating Room; An External Direct Method of Measuring the Conjugata Vera; Prolapsus Uteri without Diverticulum and with Anterior Enterocele; Lipoma of the Labium Majus; Deviations of the Rectum and Sigmoid Flexure associated with Constipation a Source of Error in Gynecological Diagnosis; Operation for the Suspension of the Retroflexed Uterus. By HOWARD A. KELLY, M. D.
Potassium Permanganate and Oxalic Acid as Germicides against the Pyogenic Cocci. By MARY SHERWOOD, M. D.
Intestinal Worms as a Complication in Abdominal Surgery. By A. L. STAVELY, M. D.
Gynecological Operations not involving Cœliotomy. By HOWARD A. KELLY, M. D. Tabulated by A. L. STAVELY, M. D.
The Employment of an Artificial Retroposition of the Uterus in covering Extensive Denuded Areas about the Pelvic Floor; Some Sources of Hemorrhage in Abdominal Pelvic Operations. By HOWARD A. KELLY, M. D.
Photography applied to Surgery. By A. S. MURRAY.
Traumatic Atresia of the Vagina with Hæmatokolpos and Hæmatometra. By HOWARD A. KELLY, M. D.
Urinalysis in Gynecology. By W. W. RUSSELL, M. D.
The Importance of employing Anæsthesia in the Diagnosis of Intra-Pelvic Gynecological Conditions. By HUNTER ROBB, M. D.
Resuscitation in Chloroform Asphyxia. By HOWARD A. KELLY, M. D.
One Hundred Cases of Ovariotomy performed on Women over Seventy Years of Age. By HOWARD A. KELLY, M. D., and MARY SHERWOOD, M. D.
Abdominal Operations performed in the Gynecological Department, from March 5, 1890, to December 17, 1892. By HOWARD A. KELLY, M. D.
Record of Deaths occurring in the Gynecological Department from June 6, 1890, to May 4, 1892.

VOLUME IV. 504 pages, 33 charts and illustrations.

Report on Typhoid Fever.

By WILLIAM OSLER, M. D., with additional papers by W. S. THAYER, M. D., and J. HEWETSON, M. D.

Report in Neurology.

Dementia Paralytica in the Negro Race; Studies in the Histology of the Liver; The Intrinsic Pulmonary Nerves in Mammalia; The Intrinsic Nerve Supply of the Cardiac Ventricles in Certain Vertebrates; The Intrinsic Nerves of the Submaxillary Gland of *Mus musculus*; The Intrinsic Nerves of the Thyroid Gland of the Dog; The Nerve Elements of the Pituitary Gland. By HENRY J. BERKLEY, M. D.

Report in Surgery.

The Results of Operations for the Cure of Cancer of the Breast, from June, 1889, to January, 1894. By W. S. HALSTED, M. D.

Report in Gynecology.

Hydrosalpinx, with a report of twenty-seven cases; Post-Operative Septic Peritonitis; Tuberculosis of the Endometrium. By T. S. CULLEN, M. B.

Report in Pathology.

Deciduoma Malignum. By J. WHITRIDGE WILLIAMS, M. D.

VOLUME V. 480 pages, with 32 charts and illustrations.

CONTENTS:

The Malarial Fevers of Baltimore. By W. S. THAYER, M. D., and J. HEWETSON, M. D.
A Study of some Fatal Cases of Malaria. By LEWELLYS F. BARKER, M. B.

Studies in Typhoid Fever.

By WILLIAM OSLER, M. D., with additional papers by G. BLUMER, M. D., SIMON FLEXNER, M. D., WALTER REED, M. D., and H. C. PARSONS, M. D.

VOLUME VI. About 500 pages, many illustrations.

Report in Neurology.

Studies on the Lesions produced by the Action of Certain Poisons on the Cortical Nerve Cell (Studies Nos. I to V). By HENRY J. BERKLEY, M. D.
Introductory.—Recent Literature on the Pathology of Diseases of the Brain by the Chromate of Silver Methods; Part I.—Alcohol Poisoning.—Experimental Lesions produced by Chronic Alcoholic Poisoning (Ethyl Alcohol). 2. Experimental Lesions produced by Acute Alcoholic Poisoning (Ethyl Alcohol); Part II.—Serum Poisoning.—Experimental Lesions induced by the Action of the Dog's Serum on the Cortical Nerve Cell; Part III.—Ricin Poisoning.—Experimental Lesions induced by Acute Ricin Poisoning. 2. Experimental Lesions induced by Chronic Ricin Poisoning; Part IV.—Hydrophobic Toxaemia.—Lesions of the Cortical Nerve Cell produced by the Toxine of Experimental Rabies; Part V.—Pathological Alterations in the Nuclei and Nucleoli of Nerve Cells from the Effects of Alcohol and Ricin Intoxication; Nerve Fibre Terminal Apparatus; Asthenic Bulbar Paralysis. By HENRY J. BERKLEY, M. D.

Report in Pathology.

Fatal Puerperal Sepsis due to the Introduction of an Elm Tent. By THOMAS S. CULLEN, M. B.
Pregnancy in a Rudimentary Uterine Horn. Rupture, Death, Probable Migration of Ovum and Spermatozoa. By THOMAS S. CULLEN, M. B., and G. L. WILKINS, M. D.
Adeno-Myoma Uteri Diffusum Benignum. By THOMAS S. CULLEN, M. B.
A Bacteriological and Anatomical Study of the Summer Diarrhœas of Infants. By WILLIAM D. BOOKER, M. D.
The Pathology of Toxalbumin Intoxications. By SIMON FLEXNER, M. D.

The price of a set bound in cloth [Vols. I-VI] of the Hospital Reports is $30.00. Vols. I, II and III are not sold separately. The price of Vols. IV, V and VI is $5.00 each.

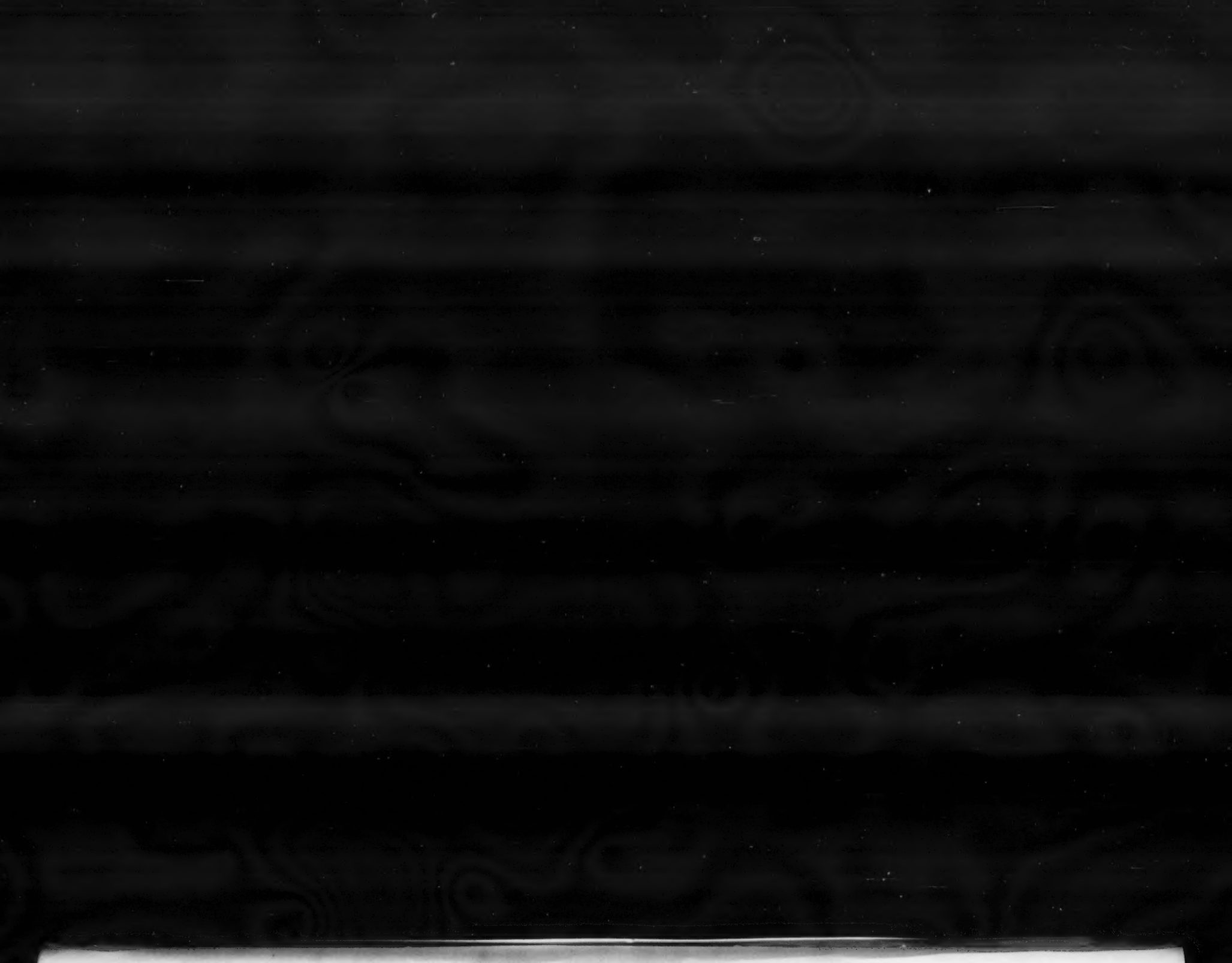
MONOGRAPHS ON DERMATOLOGY, MALARIAL FEVERS AND TYPHOID FEVER.
The following papers are reprinted from Vols. I, IV and V of the Reports, for those who desire to purchase in this form:

STUDIES IN DERMATOLOGY. By T. C. GILCHRIST, M. D., and EMMET RIXFORD, M. D. 1 volume of 164 pages and 41 full-page plates. Price, bound in paper, $3.00.

THE MALARIAL FEVERS OF BALTIMORE. By W. S. THAYER, M. D., and J. HEWETSON, M. D. And A STUDY OF SOME FATAL CASES OF MALARIA. By LEWELLYS F. BARKER, M. B. 1 volume of 280 pages. Price, in paper, $3.75.

STUDIES IN TYPHOID FEVER. By WILLIAM OSLER, M. D., and others. Extracted from Vols. IV and V of the Johns Hopkins Hospital Reports. 1 volume of 481 pages. Price, bound in paper, $3.00.

Subscriptions for the above publications may be sent to

THE JOHNS HOPKINS PRESS, BALTIMORE, MD.

The Johns Hopkins Hospital Bulletins are issued monthly. They are printed by THE FRIEDENWALD CO., Baltimore. Single copies may be procured from Messrs. CUSHING & CO. and the BALTIMORE NEWS COMPANY, Baltimore. Subscriptions, $1.00 a year, may be addressed to the publishers, THE JOHNS HOPKINS PRESS, BALTIMORE; single copies will be sent by mail for fifteen cents each.